

*Bothrops asper*, *B. atrox*, *Bothriopsis bilineata* and *Lachesis muta* cause the most severe envenoming in Ecuador. In recent years, the most widely used antivenom, 'Myn', produced by Ronti and imported from Mexico, has failed in clinical use. There is an urgent need to find an effective alternative. Four antivenoms with cover against the venoms of *Bothrops* species were compared using standard WHO rodent and *in vitro* assays (Theakston and Reid, 1983): (1) 'Myn', Ronti Mexico SA (*B. atrox*, *Crotalus durissus terrificus*, Mexico); (2) Instituto Butantan (*Bothrops* polyvalent, Brazil); (3) Instituto Nacional de Higiene y Medicina Tropical (*Bothrops* polyvalent, Ecuador); (4) Instituto Nacional de Salud (*B. asper*, *C. durissus* and *L. muta*, Colombia). The venoms against which these antivenoms were tested were Ecuadorian *B. atrox*, *B. asper* and '*B. xanthogrammus*'. Brazilian antivenom proved overall to be the most effective antivenom followed by the Ecuadorian and Colombian antivenoms. The Mexican antivenom was, in most respects, completely ineffective against the venoms of Ecuadorian *Bothrops* species. One monospecific Brazilian *L. muta* antivenom (Instituto Butantan) and the Colombian polyspecific antivenom (see above) were tested against Ecuadorian *L. muta* venom; the former was effective whereas the latter was not. Clinical trials of Brazilian, Ecuadorian and possibly Colombian antivenoms are planned in the Amazon region of Ecuador in the near future.

Theakston, R. D. G. and Reid, H. A. (1983) *Bull. WHO* 61, 949-956.

*Comparison of F(ab')<sub>2</sub> and Fab efficiency on plasma extravasation induced by Viper aspis venom.* M. Sorkine,<sup>1,2</sup> B. Salio<sup>1</sup> and C. Bon<sup>1</sup> (<sup>1</sup>Unité des Venins, Institut Pasteur 25, Rue du Dr Roux, 75724 Paris Cedex 15, France; and <sup>2</sup>S.A.R.. Hôpital Henri Mondor, Créteil 94000, France).

Envenomation caused by European vipers associates local signs, essentially oedema and systemic manifestations. Extensive oedema produces pain and inability to use the affected limb, and is a major factor of hypovolemia. Since symptomatic treatment failed to prevent this oedema, the effect of antivenom on plasma extravasation, the first step of oedema formation, was examined. The purpose of the study was to compare in a mouse model the effect of F(ab')<sub>2</sub> (equine) and Fab (equine and ovine) on capillary permeability increase (CPI) induced by *Vipera aspis aspis* venom. F(ab')<sub>2</sub> (ID<sub>50</sub> 2 mg/kg) and Fab (ID<sub>50</sub> 2.5 mg/kg) reduced considerably CPI when mixed with venom prior to intradermal injection. When fragments were intravenously injected before intradermal administration of the venom, a larger amount of fragments was necessary, Fab being five times more effective than F(ab')<sub>2</sub> (ID<sub>50</sub> 105 mg/kg compared to ID<sub>50</sub> 520 mg/kg). Furthermore, immunoglobulins injected after the venom F(ab')<sub>2</sub> were ineffective, while Fab has a residual effect (ID<sub>50</sub> 235 mg/kg). No difference was observed on the efficiency of ovine and equine Fab. These data showed firstly that the *in vitro* neutralization of the venom by immunoglobulin fragments does not reflect their *in vivo* efficiency. Secondly, Fab was considerably more effective than F(ab')<sub>2</sub> in reducing CPI induced by venom. One explanation is the different kinetics of these fragments. The smaller size of Fab results in faster diffusion and a greater volume of distribution.

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